ADENOID HYPERTROPHY PRESENTING WITH SYSTEMIC HYPERTENSION

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Abstract: *A two and half year old male child was seen with systemic hypertension, left ventricular dysfunction, mitral regurgitation and congestive cardiac failure. Examination revealed adenoid hypertrophy. He was also suffering from obstructive sleep apnea. He was being treated with anti-hypertensive and anti-failure drugs. Adenoidectomy was performed following which obstructive sleep apnea symptoms disappeared and his cardiac status improved markedly. Subsequently he was weaned off anti-hypertensive and anti-failure therapy.*

Keywords: *Obstructive Sleep Apnea (OSA), Hypertension, Adenoid hypertrophy, Left ventricular dysfunction*

INTRODUCTION:

Adenotonsillar enlargement is the most common cause for Obstructive Sleep Apnea (OSA) in children. Long standing OSA leads to hypoxia, pulmonary hypertension, right ventricular hypertrophy and corpulmonale. We report a case of severe adenoid hypertrophy causing OSA, systemic hypertension; the child had left ventricular dysfunction, mitral regurgitation and congestive cardiac failure.

CASE REPORT

A two and half year old male child presented with history of breathlessness for past one year and noisy breathing since infancy. He had history of recurrent respiratory tract infections and was unable to walk or play for long. Parents gave history of mouth breathing, snoring and disturbed short periods of sleep. His milestones were delayed. His activity was low. He was diagnosed as having myocarditis with poor ventricular function and mitral regurgitation in another hospital. Surgery for mitral regurgitation was considered in that hospital.

Examination at our hospital showed an underweight child with pallor and mouth breathing. He weighed 10 kgs [-2S.D]. He was noted to have blood pressure of 160/110 mmHg (more than 95 percentile of age, height and gender). He had dysmorphic facies and micrognathia, long low set ears with bilateral preauricular tags and pits [Fig .1]. Child had complete nasal obstruction due to postnasal mass. High arched palate was present and soft palate was bulging down.There was pansystolic murmur at apex (III & IV) with apex being displaced down and out.

At admission, Hemoglobin was - 7.3gm/dl, PCV – 26.4%, Total Count – 17.3cumm, Bun – 14mg/dl, Creatinine – 0.2mg/dl, Sodium – 136mmol/l., Potassium – 4.83mmol/l, Chloride – 96mmol/l, TSH – 8.01uI/ml, FT4 – 0.85ng/dl, FT3 – 4.34pg/dl. Chest X-ray showed 75% cardiothoraic ratio and left atrial enlargement (Fig.2). ECG showed normal axis, left ventricular hypertrophy and left atrial enlargement. Spiral CT scan showed very large adenoid mass completely occluding the nasopharnyx and airway (Fig.3). Echocardiogram showed severe left ventricular hypertrophy, moderate mitral regurgitation and ejection fraction of 50% and normal pulmonary artery pressure (Table 2). No right ventricular hypertrophy was noted . Previous echocardiograms in two other institutions showed 20% ejection fraction and 40% ejection fraction (Dimensions were not available). Abdominal and renal ultrasonogram was normal. He was on afterload reduction with enalapril,digoxin and diuretics when first seen in our hospital. A diagnosis of adenoid hypertrophy causing OSA leading to systemic hypertension with left ventricular dysfunction, mitral regurgitation and CCF was arrived. He was started on T.Envas

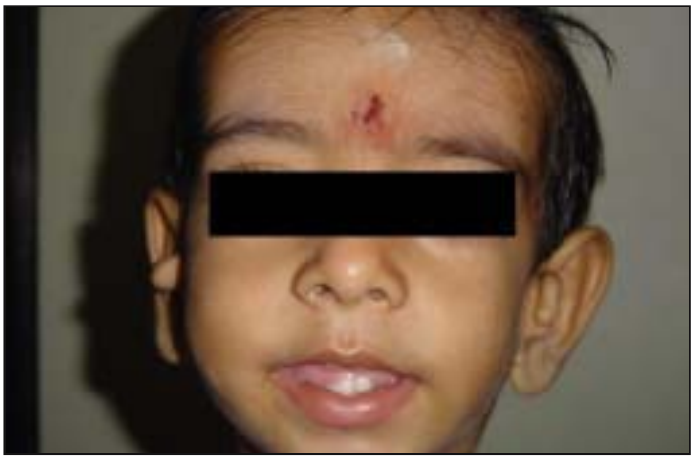

Fig.1.Patient's photograph showing dysmorphic facies, micrognathia,long low set ears with bilateral preauricular tags.

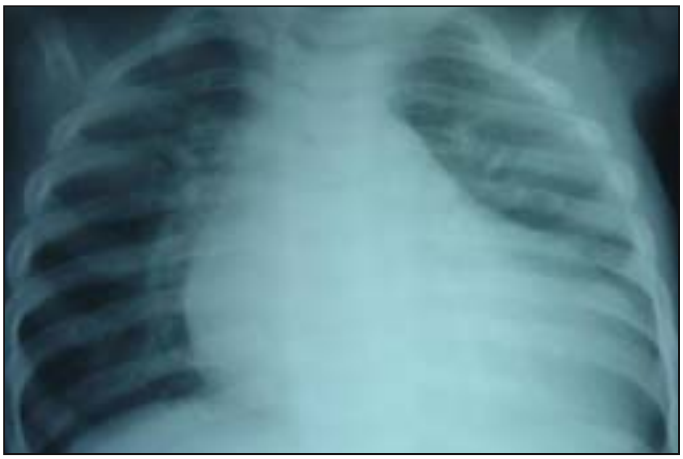

Fig.2. Pre-op chest X – ray showing increased cardiothoracic ratio and left atrial enlargement

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(Enalapril maleate) 2.5mg ½ bd, T.Depin (Nifedipine) 5mg ¼ tds,T.Fruselac (Frusemide) ¼ bd and T.Thyronorm (Thyroxine) 25mg od. Adenoidectomy under general anaesthesia was done. A very large adenoid pad was removed. No excessive bleeding in the intra and post-op period was encountered. Post-op period was uneventful. His hemodynamic status improved steadily after surgery (Table1). Anti-hypertensive dose was reduced and patient was discharged.

At review 45 days after adenoidectomy, child was very active with no evidence of nasal obstruction or OSA. He had good sleep for the first time according to his parents. His blood pressure was 90/70 mm Hg. Chest X-ray showed cardiothoraic ratio of 60% (Fig. 4). At review 8 months after adenoidectomy, child was very active and gained weight (12 kg). His blood pressure was 90/70 mm Hg. Post-op X-ray skull lateral view showed patent airway with no evidence of adenoid tissue (Fig. 5). Echocardiogram showed persisting left ventricular dilatation but with good contractility. Mitral regurgitation persisted but mild (grade I–II / IV). There was no left ventricular hypertrophy (Table 2). Ejection fraction had improved to 61%.

Table 1 – Vital parameters

Period	Pulse (Per min)	BP in mm Hg
Admission	108	160/110
Pre-OP	106	140/80
Post-OP Immediate	92	130/90
24 hrs	94	130/80
48 hrs	96	130/80
72 hrs	90	130/80
1 week	86	110/70
45 days	84	90/70

Table 2– Echo parameters

Dimensions	Pre-op	Post-op (8 months)
Left ventricular end diastolic dimension	39mm	36mm
Inter ventricular septum	10.5mm	8.0mm
Left ventricular posterior wall	9.5mm	8.0mm
Ejection fraction	50%	61%

DISCUSSION:

OSA occurs when there is a failure to maintain upper airway patency during sleep, which in turn, affects blood gas homeostasis. [1] In normal children, arterial oxygen decreases and carbon dioxide increases only slightly during sleep. But in patients with OSA these changes are severe . Decreased airflow, hypercapnia and hypoxemia are potent stimuli for increased ventilatory effort and upper airway muscle activity. Increased effort and airway muscle tone lead to resumption of airway patency . Arousal from sleep also helps in restoring blood gases. [2] Main factor producing arousal is high level of negative intra thoracic pressure generated by increased respiratory effort associated with airway obstruction. Sleep is re-established, upper airway muscle activity decreases and the cycle starts again. This cyclicity occurs in adults but is not well established in children. [1,6] Chronic hypoxemia can lead to polycythemia, growth failure, increased pulmonary artery pressure and pulmonary hypertension, right-sided heart failure, arrythmias or even death. [1] Recurrent arousals can lead to sleep fragmentation, loss of normal sleep patterns and excessive daytime sleepiness. Sleep fragmentation itself can suppress arousal responses and further impair the ability to re-establish upper airway patency and restore gas exchange. [1,4,5] The threshold for arousal is elevated during REM sleep such that obstructive episodes are more prolonged and severe during this sleep stage. Studies also show that patients with cranio facial abnormalities are prone for OSA.

[2,3] In case of adenoid hypertrophy there is upper airway narrowing which results in impaired gas exchange with hypoxemia and hypercapnia. Hypoxia is associated with a rise in sympathetic output and catecholamine production and the resulting peripheral vasoconstriction causes transient pulmonary and systemic hypertension. Hypoxia can cause a variety of cardiac arrhythmias, which range from bradycardias to ventricular ectopics.

CONCLUSION :

This case highlights the need for increased awareness among pediatricians and cardiologists about the possibility of OSA causing pulmonary and systemic hypertension in children. A common pediatric problem like adenoid hypertrophy could

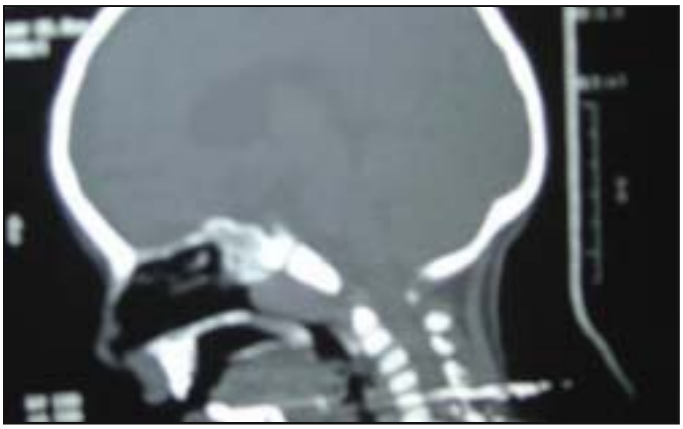

Fig.3. Spiral CT scan showing adenoid hypertrophy obstructing the airway

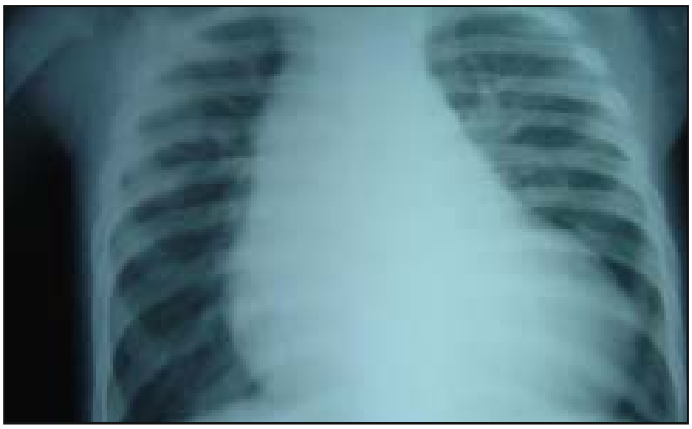

Fig.4. Post -op chest X – ray showing significant decrease in cardiothoracic ratio

possibly lead to serious complications like severe systemic hypertension, left ventricular dysfunction, mitral regurgitation and CCF.

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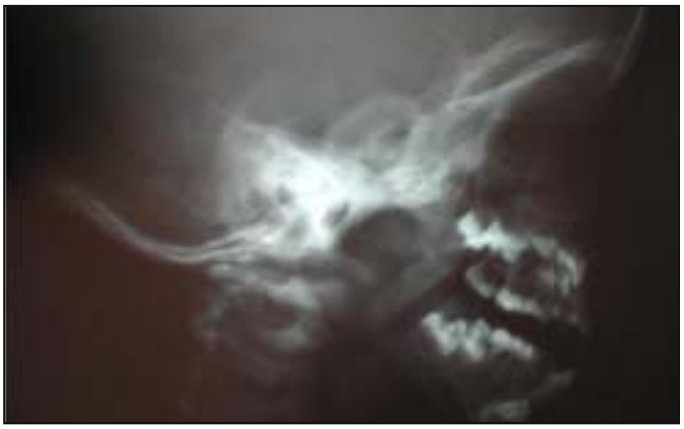

Fig.5 . Post-op lateral view of X-ray skull shows patent airway following adenoidectomy

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